centres served by the Red Cross and its volunteer donors, been so readily available that use has become abuse. I am certain that well over half of the blood now being used is used unnecessarily. This is wasteful. Even more, it is dangerous, and in this series of articles I have tried to indicate some of the dangers; I have said nothing about the danger of hepatitis following whole blood transfusion or about certain other dangers, but have limited myself to some sequelæ of blood group sensitization. Second, when the Red Cross undertook to develop a free national transfusion service, it took from most hospitals, and from most universities too, a first-hand knowledge of immunohæmatology. With certain exceptions our hospitals and universities not only have not added to our knowledge in this field but have not even kept up with the new knowledge that has developed in the past 17 years. This is serious. It has not only retarded advance of knowledge but has rendered many hospitals technically incompetent in the field of blood transfusion; in at least three of the above cases the reactions were due to errors of blood grouping or cross-matching technique.

What can be done about all this? The problem of over-transfusion can be solved only by the profession. The responsibility devolves upon the individual physician and upon the organized hospital staffs.

Personally I should like to see the transfusion service become a co-operative effort shared by the Red Cross and the hospitals, the Red Cross supplying most of the donor blood but the hospital being responsible for typing, matching and the use of blood and, where desired, for at least a part of the donor blood. Such an arrangement would require that the blood bank and transfusion service in the larger hospitals be in charge of a competent and responsible medical director provided with an adequate staff and given reasonable authority over the issuing of blood; smaller hospitals might well form a liaison with the larger ones, might in essence become branch offices of the blood banking system, and have their personnel trained and kept up-to-date by the parent bank.

There is a drawback; any such arrangement will cost money. The costs would presumably be hospital costs and would be included in hospital rates, whether the rates were paid on a personal basis or through hospital insurance—Blue Cross, commercial or state. I can see no other way out of our present dilemma.

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RADIATION DAMAGE CAUSED BY SHOE-FITTING FLUOROSCOPE

A case of radiodermatitis occurred on the dorsum of the right foot of a 56-year-old woman who had worked for 10 years in a shoe shop where a fluoroscope was used for shoe-fitting. It is presumed that only one foot was involved because of a defect in the lead shielding which allowed the operator to be exposed 10 or 15 minutes a day to a considerable dosage of x-radiation. It was also determined that there was abdominal exposure of about 1 r per minute in adults. In children this would have been much higher.—H. Kopp: *Brit. M. J.*, 2: 1344, 1957.

SHORT COMMUNICATION

CUBITAL TUNNEL COMPRESSION IN TARDY ULNAR PALSY

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OVER THE PAST TWO YEARS we have been prompted to reassess the anatomical factors involved in tardy ulnar palsy, because of the surgical findings in three patients who had no significant degree of distortion of the elbow joint or of the ulnar groove.

CLINICAL FINDINGS

In two patients there was a history of injury to the elbow but this had resulted in only a mild valgus deformity. The other patient gave no history of trauma. Otherwise, in all three patients the clinical picture was remarkably similar. They presented with the classical symptoms of coldness, numbness or tingling, followed by muscle weakness and wasting in the ulnar distribution. A prominent complaint was aching pain along the ulnar border of the forearm and hand. On palpation the nerve in the ulnar groove was swollen and insensitive. A significant sign was the sparing or minimal weakness of the flexor carpi ulnaris muscle, as contrasted with the marked loss of power and wasting in the remaining muscles supplied by the ulnar nerve. This distinction was also borne out by electrical studies.

OPERATIVE FINDINGS

The first two cases were accepted as classical examples of tardy ulnar palsy as described by Panas[7] in 1878. The nerve was exposed to carry out an anterior transportation in the usual manner in order to eliminate the friction and stretching which generally have been considered as the causative factors in the production of the paralysis.[1, 2] As a first stage in this procedure, the aponeurosis joining the two heads of the flexor carpi ulnaris muscle was incised as it arches between the medial epicondyle and the olecranon. It was then noted that the nerve was constricted at the point where it dips beneath this aponeurotic arch and was swollen proximal to it. In one case, deep to the aponeurosis there was a fibrous membrane arching in the same direction which appeared also to be contributing to the constriction of the nerve.

In both cases, the nerve was freed of adhesions in the ulnar groove and this aponeurotic arch over the nerve was slit open. The nerve was not transposed. Nevertheless, prompt relief of pain with immediate improvement of numbness and motor

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Canad. M. A. J.
Mar. 1, 1958, vol. 78

power followed this procedure—in one patient, pain sensibility improved as soon as the brachial plexus anæsthetic block had worn off. In a third patient, treated in the same manner, the flexion deformity of the fourth and fifth fingers was no longer apparent three hours after operation. The first patient was operated upon in April 1956, and the others in October 1956 and January 1957. Since on repeated postoperative reviews they have shown continuing improvement with return of useful hand function, it now seems worth while to direct wider attention to this interesting problem.

THE CUBITAL TUNNEL

The focal constriction and retrograde œdema of the nerve, and the striking promptness of improvement following simple decompression of the nerve, have led us to the inescapable conclusion that an important factor in the causation of tardy ulnar palsy, and one which seems to have escaped previous analysis, is compression of the nerve just distal to the ulnar groove. At this point the nerve dips from a superficial to a deep submuscular course through a restricted opening. The roof of this opening is formed by the aponeurotic arch stretching between the medial epicondyle and the olecranon where the two heads of the flexor carpi ulnaris are attached. The floor is formed by the medial ligaments of the elbow joint. Because of the analogy in some respects to the nerve compression within the carpal tunnel, it has been proposed[3] that this opening be called the cubital tunnel.*

THE EFFECTS OF THE CUBITAL TUNNEL

This tunnel offers an anatomical basis for some of the features of tardy ulnar palsy which have not been satisfactorily explained previously. These include the soft swelling of the nerve so commonly observed at operation and which is so distinct from the firm scar tissue of a true neuroma. We suggest that such constriction causes œdema of the nerve by interruption of the proximo-distal flow along the nerve or by compression of the longitudinally arranged blood vessels within the nerve.

Secondly, the frequent sparing of the flexor carpi ulnaris muscle may be related to the fact that this muscle, of all those supplied by the ulnar nerve, is the only one which has motor nerve branches separating off from the main nerve proximal to the tunnel. It seems probable that these branches, being smaller and lying somewhat more freely in the tunnel, are less subject to compression than the larger bulk of the main ulnar nerve. In our experience the sparing of this muscle is a distinctive sign of cubital funnel compression.

Thirdly, the well-recognized aggravation of symptoms by flexion of the elbow, which occurred in the cases reported here during milking, shovelling or hammering, can be attributed to narrowing of the cubital tunnel during such movement. Previously, it had been attributed to stretching and friction of the nerve in the presence of cubitus valgus. But, as in our cases, valgus deformity is absent or of minimal significance in many patients with tardy ulnar palsy. Moreover, in the cases noted here, recovery occurred without shortening the anatomical course of the nerve. Examination of the elbow by careful palpation and during exposure at operation indicates that during flexion of the limb several factors act to produce this narrowing of the cubital tunnel. Firstly, the roofing aponeurosis becomes taut as its point of attachment to the olecranon moves further forward and away from its fixed attachment to the medial epicondyle. Secondly, the floor of the tunnel is elevated because the medial ligament of the elbow joint bulges outward during flexion. This not only further narrows the tunnel but also contributes a third factor—an increase in the angle of dip to which the nerve is subjected as it enters the tunnel from its superficial course in the ulnar groove. All three factors act at a maximum when the limb is in flexion. This narrowing of the tunnel when the elbow is bent would also explain the "sleep paralysis" of the ulnar nerve when the limb is kept in prolonged flexion, without external pressure, as noted in three cases by Gowers.[4]

A finding observed in our first case was the presence of a connective tissue band crossing the nerve just distal to the ulnar groove and deep to the fibrous arch of the aponeurosis. It contributed to the nerve compression by enhancing the constricting effect of the cubital tunnel. A similar but less well developed band present in the third case did not appear to be adding much to the effect of the cubital tunnel. In other dissections of the elbow, we have noted that such bands may be well developed or absent, and we therefore do not consider their presence necessary to the development of cubital tunnel compression. There is no doubt, however, that such accessory bands, when present, must also be slit open with the roofing aponeurotic arch, or the nerve will be inadequately decompressed.

Since we reported these findings,[3] our attention has been directed to the abstract of an interesting preliminary communication by Osborne.[5] In this he reports that in exploring cases of tardy ulnar neuritis he had found in almost every case a band of fibrous tissue bridging the two heads of the flexor carpi ulnaris. He considered this band responsible for compression of the nerve. It is not clear whether this refers to the aponeurosis of the flexor carpi ulnaris, which is a constant and well-recognized anatomical structure* and which in our

*From Latin *cubitum* = elbow. *Cubital* was used by Horace in the sense of elbow cushion, suggesting that the Romans were not unaware of the hazard to the ulnar nerve presented by reclining banquets!

*For example, Cunningham's *Manual of Practical Anatomy* (1935, p. 143) refers to this aponeurosis as a "fibrous arch between the heads of the flexor carpi ulnaris".

view forms the important roofing component of the cubital tunnel; or whether it refers to the deeper fibrous bands, noted previously by Woltman,[6] which are an accessory anatomical feature making in our cases a variable contribution to the nerve constriction. The description of tightening of the band with flexion of the limb applies equally well, of course, to either structure.

But both of these structures overlying the nerve are less meaningful if not considered in relation to the ligamentous floor of the tunnel which offers counter-pressure by bulging during flexion. Moreover, it may be noted that one of the patients reported by Panas[7] in 1878, and a considerable proportion of the patients in larger series of cases published since then, were described only as having arthritis with no history of trauma to the elbow. It is suggested that the puzzling progress of palsy in these cases can be explained by the scarring of the ligamentous joint tissue, thus producing thickening of the floor of the cubital tunnel with resultant nerve compression.

We consider it useful to call this particular type of tardy ulnar palsy the cubital tunnel syndrome. This term serves to indicate a focal constrictive lesion of the nerve (similar in some respects to the carpal tunnel syndrome) which tends to spare the flexor carpi ulnaris and which is relieved by simple decompression. This clinical picture is thereby distinguished from those cases of tardy ulnar palsy where paralysis of all the components of the nerve is associated with fracturing or gross distortion of the joint due to trauma. It is for this latter condition that the older term of traumatic ulnar neuritis (Platt[8]) might perhaps be reserved, and for which anterior transposition of the nerve may still be indicated. It appears probable that in many cases the successful result attributed to transposition may have been an unrecognized benefit of incidentally decompressing the cubital tunnel. If cubital tunnel decompression alone had been done, an equally satisfactory result may well have been produced.

The electromyographic findings in these cases were reported at the annual scientific meeting of the Canadian Asociation of Physical Medicine and Rehabilitation in Toronto on June 21, 1957, by our associate Dr. Talmage Hunt. Detailed case reports and a full discussion of the surgical findings are being published in the *Canadian Journal of Surgery*.

SUMMARY

In three patients, tardy ulnar nerve palsy was relieved simply by incising the aponeurotic arch overlying the nerve between the bony attachments to the olecranon and medial epicondyle of the two heads of the flexor carpi ulnaris. The nerve was seen to be constricted at this point and swollen proximal to it. There was prompt and continuing improvement after such decompression.

The term cubital tunnel is proposed for this opening through which the nerve passes from its superficial course to its deep submuscular plane just distal to the elbow. The roof of the tunnel is formed by the aponeurosis of the flexor carpi ulnaris and the floor by the medial ligament of the elbow joint. During flexion of the elbow, the tunnel is narrowed because of the tightening of the aponeurotic roof and the bulging of the ligamentous floor.

It is submitted that the cubital tunnel explains the constriction and retrograde swelling of the nerve, the aggravation of symptoms by flexion of the limb, and the sparing of the flexor carpi ulnaris muscle. These features, in the absence of gross distortion of the elbow joint, make up a typical picture which we feel may be termed the cubital tunnel syndrome.

It is suggested that the successful results obtained by anterior transposition of the ulnar nerve may have been due in many cases to the fact that a preliminary stage in this procedure incidentally involves opening the cubital tunnel and decompressing the nerve.

It is hoped that this record of our brief series of cases will stimulate interest in this problem and in the evaluation of the role of the cubital tunnel in ulnar nerve lesions.

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AORTIC COMMISSUROTOMY: A PHYSIOLOGICAL EVALUATION BY COMBINED HEART CATHETERIZATION

Data from combined left and right heart catheterization are presented by Smith *et al.* (*J. Thoracic Surg.*, 34: 815, 1957) on 14 patients with aortic stenosis, before and after surgery. The effect of aortic commissurotomy on the pathologic physiology of this lesion is described. The failure of the altered physiology to be restored completely to normal by aortic commissurotomy is pointed out and certain reasons for it are suggested. These are: (1) the inability of existing surgical techniques completely to restore the aortic valve to normal function, and (2) the secondary pathologic changes which have taken place in the myocardium of the left ventricle and atrium as a result of long-term obstruction. While aortic commissurotomy fails to restore the circulatory hæmodynamics to normal in the majority of persons, the symptom triad—angina, syncope, and dyspnœa—is relieved in many cases. The rationale for this relief is discussed. Early surgical relief of aortic stenosis is suggested in order to avoid advanced secondary valvular deformation and irreversible myocardial damage.

It appears that with the recent developments of open heart surgery, direct vision technique not only may permit much more adequate commissurotomy, but offers hope that replacement of the valve will some day be possible in severely calcified cases.